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	L3	INSULIN adj GROWTH adj FACTOR adj BINDING adj PROTEIN adj 2 OR IGFBP adj 2	. 27
	DB=PC	SPB, USPT; PLUR = YES; OP = OR	
$\Gamma$	L2	L1 and (K180A or K181A or K227A or K234A or K237A or 114 adj 170)	1
Γ	L1	INSULIN adj GROWTH adj FACTOR adj BINDING adj PROTEIN adj 2 OR IGFBP adj 2	326

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=> s insulin(w)growth(w)factor(w)binding(w)protein(w)2 or IGFBP(w)2

6166 INSULIN(W) GROWTH(W) FACTOR(W) BINDING(W) PROTEIN(W) 2 OR IGFBP( Ll W) 2

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1 L1 AND (K180A OR K181A OR K227A OR K234A OR K237A OR 114(W) 170)

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ANSWER 1 OF 1 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER:

2004:60547 CAPLUS

DOCUMENT NUMBER:

140:105836

TITLE:

Characterization and therapeutic uses of altered

insulin-like growth factor binding proteins '

INVENTOR(S):

Forbes, Briony

PATENT ASSIGNEE(S):

The University of Adelaide, Australia

SOURCE:

PCT Int. Appl., 43 pp. CODEN: PIXXD2

DOCUMENT TYPE:

Patent

LANGUAGE:

English

FAMILY ACC. NUM. COUNT:

PATENT INFORMATION:

PATENT NO.					KINI	KIND DATE		APPLICATION NO.						DATE			
WO 2004007543					A1	20040122		WO 2003-AU898						20030711			
	W:	ΑE,	AG,	AL,	AM,	AT,	ΑU,	ΑZ,	BA,	BB,	BG,	BR,	BY,	ΒZ,	CA,	CH,	CN,
		CO,	CR,	CU,	CZ,	DE,	DK,	DM,	DZ,	EC,	EE,	ES,	FI,	GB,	GD,	GE,	GH,
		GM,	HR,	HU,	ID,	IL,	IN,	IS,	JP,	KE,	KG,	ΚP,	KR,	ΚZ,	LC,	LK,	LR,
		LS,	LT,	LU,	LV,	MA,	MD,	MG,	MK,	MN,	MW,	MX,	MZ,	NI,	NO,	NZ,	OM,
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		TR,	TT,	TZ,	UA,	UG,	US,	UZ,	VC,	VN,	YU,	ZA,	ZM,	ZW			
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		BF,	ВJ,	CF,	CG,	CI,	CM,	ĠA,	GN,	GQ,	GW,	ML,	MR,	NE,	SN,	TD,	TG
CA 2491917					A1		20040122 CA 2003-2491917						20030711				
AU 2003243829 .					A1		20040202 AU 2003-243829					20030711					
EP 1534744					A1		2005	20050601 EP 2003-763510				2	20030711				
	R:	AΤ,	BE,	CH,	DE,	DK,	ES,	FR,	GB,	GR,	IT,	LI,	LU,	NL,	SE,	MC,	PT,

IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK

JP 2006514535 T 20060511 JP 2004-520184 20030711

US 2006153853 A1 20060713 US 2004-519890 20041229

PRIORITY APPLN. INFO.: AU 2002-950188 A 20020712

WO 2003-AU898 W 20030711

AB Altered IGFBPs are able to bind IGF but the release is inhibited by resistance to protease cleavage and/or reduced binding to extracellular matrix (ECM). Alterations have been made in IGFBP-2

to the linker domain in particular and two amino acid motifs found to be important for ECM binding. IGF-1 mediated proliferation of cancer cells have been inhibited by the use of these altered IGFBPs.

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